Marriage to a Smoker and Lung Cancer Risk

CHARLES G. HUMBLE, MS, JONATHAN M. SAMET, MD, MS, AND DOROTHY R. PATHAK, PHD, MS

Abstract: As part of a population-based case-control study of lung cancer in New Mexico, we have collected data on spouses' tobacco smoking habits and on-the-job exposure to asbestos. The present analyses include 609 cases and 781 controls with known passive and personal smoking status, of whom 28 were lifelong nonsmokers with lung cancer. While no effect of spouse cigarette smoking was found among current or former smokers, never smokers married to smokers had about a two-fold increased risk of lung cancer. Lung cancer risk in never smokers also increased with duration of exposure to a smoking spouse, but not with increasing number of cigarettes smoked per day by the spouse. Our findings are consistent with previous reports of elevated risk for lung cancer among never smokers living with a spouse who smokes cigarettes. (*Am J Public Health* 1987; 77:598–602.)

Introduction

The causal association of active cigarette smoking with lung cancer has been accepted for many years.[1,2] Recent epidemiologic evidence indicates that involuntary exposure of nonsmokers to tobacco smoke is also associated with lung cancer.[3–5] Nonsmokers, as well as active cigarette smokers, inhale environmental tobacco smoke, which consists of a combination of sidestream smoke and exhaled mainstream smoke. The putative association of environmental tobacco smoke with lung cancer derives biological plausibility from the lack of a demonstrated threshold for lung cancer in active smokers, from the qualitative similarities of mainstream and sidestream smoke, and from the presence of mutagens in the urine of passive smokers.[5,6]

The association of involuntary exposure to tobacco smoke with lung cancer has now been examined in studies conducted in Japan, Greece, Hong Kong, Scotland, Germany, and the United States.[5] These studies generally indicate an increased risk in nonsmokers. Studies from Japan, Greece, and the United States have shown elevated risk estimates associated with the exposure of nonsmokers to their spouses' smoking.[3,4,7–10] Increased risks have not been found in all investigations, although estimates of effect from those reports with negative findings are generally consistent with those from reports showing elevated risks.[11–16]

In 1980 we began collecting data in a population-based case-control study designed to explain differing lung cancer occurrence in Hispanic and non-Hispanic Whites in New Mexico.[17] The original study questionnaire included questions on tobacco smoke exposure from spouse smoking and on indirect exposure to asbestos through a spouse's job. This report describes the risks associated with these exposures in smokers and nonsmokers in New Mexico.

Methods

Case Selection

The cases were Hispanic and non-Hispanic residents of New Mexico, less than 85 years of age at diagnosis of primary lung cancer. Cases were ascertained by the New Mexico Tumor Registry, a member of the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute.[18] An initial case series was selected from patients with cancer incident between January 1, 1980 and December 31, 1982. For this initial series all cases less than 50 years of age and all Hispanics were included; non-Hispanics age 50 or older were sampled randomly to select 40 per cent of the males and 50 per cent of the females. To increase the size of the female non-Hispanic subgroup and Hispanics of both sexes, we selected additional cases: all patients in these groups with cancer incident between December 1, 1983 and November 30, 1984. Of the 724 eligible cases selected for the study, interviews were completed with 641, or 88.5 per cent. Of the interviews with cases, 305 were completed with the cases themselves and 336 were with surrogates, generally either the surviving spouse or a child.

For the cases in nonsmokers, the histopathological type of lung cancer was classified by panel review of histopathological material (N = 17) or by information in the New Mexico Tumor Registry case abstract (N = 28). The panel, which included two pathologists, determined the histopathological type on the basis of conventional light microscopy and used a modification of the World Health Organization classification.[19,20]

Control Selection

Potential controls were ascertained by two methods. Residences, identified from lists of randomly generated telephone numbers, were called and a household census was taken from the person who answered. Telephone sampling identified 2,038 potentially eligible households, of which 287 (14.2 per cent) refused to cooperate with the census. As this technique was not efficient for selecting older controls, an additional 252 persons were chosen from a list of randomly selected New Mexico residents, 65 years and older, who were on the Health Care Financing Administration's roster of Medicare participants. The control group was frequency-matched to the cases for sex, ethnicity, and 10-year age category at a ratio of approximately 1.2 controls per case. Of the 944 controls selected for this study, 784 (83.1 per cent) were interviewed.

Interview Data Collection

The interviews were conducted by bilingual interviewers. Respondents were asked to describe the smoking habits of all spouses of the index subject. For each smoking spouse, duration of use and average amount smoked daily were recorded for cigarettes, cigars, and pipes. Respondents were not asked to describe exposures to tobacco smoke at work or in other situations outside of the home. All jobs held by a spouse for one year or more also were recorded, as were reports of spouses' on-the-job exposures to arsenic, asbestos, lead, pesticides, and radiation. We hypothesized a priori that asbestos exposure might increase lung cancer risk and

From the New Mexico Tumor Registry, the Departments of Medicine and of Family Community and Emergency Medicine, and the Interdepartmental Program in Epidemiology, University of New Mexico Medical Center, Albuquerque. Address reprint requests to Jonathan M. Samet, MD, New Mexico Tumor Registry, University of New Mexico Medical Center, 900 Camino de Salud NE, Albuquerque, NM 87131. This paper, submitted to the Journal July 18, 1986, was revised and accepted for publication November 17, 1986.

added the other agents to reduce the emphasis on asbestos and to test for information bias. A detailed history of personal cigarette use was collected from subjects who had smoked for six months or more.

Calculation of Passive Exposure Indices

Measures of passive exposure to tobacco smoke and to asbestos were created by summarizing the information provided for each spouse. For tobacco smoke, categorical and continuous measures of exposure were calculated. We designated as "exposed" subjects ever married to a spouse who smoked cigarettes, regardless of the spouse's use of pipes or cigars. To examine the effects of cigarette smoke alone, subjects whose spouses had smoked other tobacco products were excluded from some analyses. We created two indicator variables for these exposures: one for all forms of tobacco smoke, and the other for cigarette smoke alone. We also calculated the duration of exposure to a cigarette-smoking spouse and the average number of cigarettes smoked daily by the spouse(s). If complete data were unavailable for all marriage partners, these variables were set to unknown.

Two categorical variables were created to describe potential indirect exposure to asbestos through a spouse's job. Spouse's job histories were reviewed against a list of jobs judged a priori as possibly involving exposure to asbestos: asbestos mining, textile manufacturing, auto brake repair, cement or construction work, pipe fitting or covering, insulation work, and shipyard work. If one or more jobs held by the spouse appeared on the list, the index subject was classified as exposed. Similarly, if a spouse was described as exposed at work to asbestos the index subject was considered to be exposed.

Data Analysis

For these analyses, cigarette smokers were those individuals who had smoked at least six months. Current smokers were those still smoking at interview or who had stopped within the previous 18 months; ex-smokers had ceased smoking at least 18 months before interview. The status of cases classified by questionnaire as never smokers was verified against hospital chart summaries on file at the New Mexico Tumor Registry. Of the 28 reported nonsmokers, the summaries showed that three cases had smoked cigarettes and that one case had smoked pipes and cigars regularly. Analyses of the data for never smokers were performed with and without these four subjects. Because the study included only eight males who had never smoked cigarettes, all analyses were performed for females alone and for all subjects combined.

We used the Mantel-Haenszel technique to control for ethnicity and age in estimating odds ratios for passive exposure to cigarette smoke, within strata of personal cigarette smoking.[21] In these analyses, age was categorized as below 65 years or 65 years and greater. Among never smokers, the exposure-response relation of lung cancer risk with average cigarettes smoked daily by the spouse and with duration of passive cigarette exposure was tested using Mantel extension methods for stratified data.[22] For these variables, strata of exposure were defined by the median level among all exposed never smokers. Those never exposed were the reference group for all analyses.

To examine further the effects of the passive exposures, logistic regression models were fitted for smokers and never smokers. All models included adjustment for ethnicity and four categories of age, variables for which the controls had been frequency matched to the cases. In the model for smokers, potential confounding by personal cigarette use was controlled by entering the average daily cigarette consumption, the duration of smoking, years since stopping for ex-smokers, and an interaction term calculated as the product of smoking duration and an indicator variable for age less than 65 years or 65 years and older. This model was selected on the basis of analyses described in more detail elsewhere.[23] The all-subjects models included control for sex. The two categorical indicators of passive exposure were tested individually in each model. Trends in risk with number of cigarettes of exposure daily and with duration were examined by fitting models with indicator variables to define categories of unexposed, exposed at or below the median, and above the median.

TABLE 1—Sex, Ethnicity, and Age Distribution of Subjects by Personal Cigarette Smoking Status in a Case-Control Study in New Mexico, 1980–84

Subjects	Age (years)	Cigarette Smoking Status: Current Case	Current Control	Former Case	Former Control	Never Case	Never Control
Male							
Hispanic							
White	<65	34	22	10	18	0	10
	≥65	47	30	27	29	1	21
Non-Hispanic							
White	<65	77	57	19	59	1	36
	≥65	82	60	62	103	6	63
Female							
Hispanic							
White	<65	11	8	3	7	2	27
	≥65	27	6	5	5	7	34
Non-Hispanic							
White	<65	74	34	8	17	3	47
	≥65	64	15	31	19	8	54

Risk estimation for the effect of indirect exposure to asbestos was limited to females as no males were indirectly exposed. Logistic regression models were employed that controlled for active smoking as described above, for current and ex-smokers, and for marriage to a smoker for never smokers.

Because surrogate interviews were necessary for 52 per cent of the cases, we assessed the effect of information source by performing the analyses separately for self-reported and surrogate-reported cases, using self-reported controls. We excluded from these analyses the 13 controls for whom surrogate interviews had been necessary.

All cross tabulations and logistic models were performed with standard programs of the Statistical Analysis System.[24] Odds ratios (OR) and 90 per cent two-sided Cornfield confidence intervals (CI) were calculated using program 23 from the Rothman and Boice text for programmable calculators.[25,26]

Results

The analyses were restricted to those 1,390 subjects with known passive and personal smoking status (Table 1). The 35 excluded subjects were older than those included (mean age 68.4 vs 65.6 years, respectively). More cases were excluded than controls (5.0 per cent vs 0.4 per cent, respectively), due in part to the greater proportion of surrogate interviews for cases than for controls. The percentage of subjects excluded did not differ by ethnicity or sex.

Based on data in the New Mexico Tumor Registry files, the cases described by interview data as "never smokers"

TABLE 2—Odds Ratio[a] Estimates for Passive Cigarette Exposure in a Case-Control Study of Lung Cancer in New Mexico, 1980–84

Passive Exposure	Personal Smoking Status	All Subjects OR	All Subjects 90% CI[b]	Females Only OR	Females Only 90% CI[b]
Cigarettes only	Current	1.2	0.9, 1.6	0.9	0.4, 2.2
	Former	1.1	0.8, 1.5	0.7	0.2, 2.2
	Never	2.9	1.3, 6.7	1.8	0.6, 5.4
Cigarettes and/or pipe or cigar	Current	1.2	0.9, 1.6	0.9	0.5, 1.8
	Former	1.1	0.8, 1.5	0.8	0.2, 1.7
	Never	3.2	1.5, 7.2	2.3	0.9, 6.6

[a]From crude cross tabulations; adjustment for age or for ethnicity did not alter results.
[b]Two-sided 90 per cent Cornfield confidence intervals.

TABLE 3—Odds Ratio Estimates from Multiple Logistic[a] Analyses of Passive Cigarette Exposure and Lung Cancer Risk, in a Case-Control Study in New Mexico, 1980–84

Passive Exposure	Personal Smoking Status	All Subjects OR	All Subjects 90% CI	Female Only OR	Female Only 90% CI
Cigarette only	Ever[b]	1.0	0.8, 1.4	1.0	0.5, 1.9
	Never	2.2	1.0, 4.9	1.7	0.6, 4.3
Cigarettes and/or pipe or cigar	Ever[b]	1.0	0.8, 1.3	0.9	0.5, 1.5
	Never	2.6	1.2, 5.6	2.2	0.9, 5.5

[a]All models included variables to control for the frequency matching on age and ethnicity, and sex, when appropriate.
[b]Models for smokers controlled for personal cigarette use as described under Methods.

TABLE 4—Odds Ratio[a] Estimates by Duration of Spouse Cigarette Smoking and by Average Cigarettes Smoked Daily by the Spouse(s) among Never Smokers in a Case-Control Study in New Mexico, 1980–84

Subject Group	Duration ≤26 Years OR	Duration ≤26 Years 90% CI	Duration >26 Years OR	Duration >26 Years 90% CI	Chi for trend
All Subjects	2.2	0.8, 5.9	2.7	1.0, 7.1	2.01
Females only	1.6	0.5, 5.8	2.1	0.7, 6.9	1.23
	Mean Cigarettes per Day ≤20 OR	Mean Cigarettes per Day ≤20 90% CI	Mean Cigarettes per Day >20 OR	Mean Cigarettes per Day >20 90% CI	
All Subjects	2.8	1.2, 6.6	2.2	0.6, 7.3	1.82
Females only	1.8	0.6, 5.6	1.2	0.3, 5.2	0.46

[a]Odds ratios not adjusted for age or ethnicity. Adjustment for either of these factors did not change the results. The referent category was the never exposed.

who were ever married to a smoking spouse included eight adenocarcinomas, two epidermoid carcinomas, two small cell carcinomas, and four large cell carcinomas. The eight nonexposed cases reported to be never smokers comprised six adenocarcinomas and two epidermoid carcinomas. A specific histological type had not been assigned to four of the cases. Of the four cases in reported never smokers but who were identified by Tumor Registry information as smokers, one was small cell carcinoma, two were adenocarcinoma, and one was not classified. Because material was only retrieved for 17 cases for panel review, we did not compare the exposed and nonexposed based on the pathologists' classification. Of the 17 cases, the cell type based on the panel's review concurred with that in the Registry for only eight cases.

In the never smoking controls, marriage to a smoker of any type of tobacco was reported for 28 per cent of males and for 56 per cent of females. The corresponding percentages for marriage to a smoker of cigarettes alone were similar, 28 per cent for males and 57 per cent for females.

Using stratified and unstratified approaches, no effect of marriage to a smoker was found among current or former cigarette smokers (Table 2). By contrast, among never smokers, cigarette smoking by a spouse, regardless of pipe or cigar use, was associated with a three-fold increased risk of lung cancer. Adjustment for ethnicity (OR = 3.2, 90 per cent CI [Confidence Interval] = 1.5, 7.2) or for age (OR = 3.2, 90 per cent CI = 1.5, 7.3) did not change the estimated risks. A similar close agreement of crude (Table 2) and adjusted estimates was observed for exposure to cigarettes only: ethnicity-adjusted OR = 3.0 (CI = 1.3, 6.8) and age-adjusted OR = 2.9 (CI = 1.3, 6.7). There were insufficient subjects to adjust simultaneously for ethnicity and age. Although the odds ratios were reduced, restriction of the sample to females did not change the pattern of effect from that found in the analyses with all subjects. When the analyses were performed separately for self- and surrogate-reported cases, the odds ratios were comparably elevated for both groups (data not shown). Because the control series did not include sufficient numbers of controls with surrogate interviews, the controls could not be similarly stratified by type of interview.

Odds ratios from the logistic models (Table 3) tended to be lower than from the unstratified and stratified analyses (Table 2). Risk estimates for the current and former smokers from the logistic models also showed no effect of passive cigarette exposure beyond that of active smoking. However, among the never smokers all point estimates were above unity.

Assessment of exposure-response relation for the duration of exposure and for the average cigarettes smoked daily by the spouse was limited to never smokers. For the all-subjects and females-only cross tabular analyses, a pattern of increased risk with greater duration of cigarette exposure was found (Table 4). In contrast, the logistic models did not show an increase with duration of exposure in either group: (for all subjects, short duration OR = 1.9, CI = 0.7, 4.7; long duration OR = 1.8, CI = 0.7, 4.5). The exposure-response pattern for cigarettes smoked daily showed higher odds ratios for subjects whose spouses smoked a pack or less per day than for those whose spouses smoked greater amounts (Table 4). Control of stratification factors by multiple logistic modeling did not change the pattern of higher relative risk estimates for nonsmokers exposed to 20 or fewer cigarettes per day (OR = 2.0, CI = 0.9, 4.6) compared with those exposed at higher levels (OR = 1.6, CI = 0.5, 4.9). The respective logistic estimates for females were lower: OR for daily exposure of 20 cigarettes or less was 1.6 (CI = 0.6, 4.3) while for exposure to more than 20 cigarettes the OR was 1.2 (CI = 0.3, 4.4).

Potential indirect exposure to asbestos was only reported for females. In the controls, 14.5 per cent of women were designated as exposed based on their husband's work history and 8.2 per cent were considered as exposed based on a report of their husband's occupational exposure to asbestos. The effects of the asbestos exposure variables were assessed

TABLE 5—Estimates of Lung Cancer Risk from Spouse's Occupational Exposure to Asbestos, by Reporting Source, for Females in a Case-Control Study in New Mexico, 1980–84

Personal Smoking Status		Employment in Asbestos-Related Job		
		All Subjects	Self-reported	Surrogate[b] reported
Ever[a]	OR	0.8	0.7	1.1
	90% CI	0.4, 1.6	0.3, 1.5	0.5, 2.8
Never	OR	2.5	1.2	3.3
	90% CI	1.0, 6.4	0.2, 8.2	1.1, 9.5
		Reported as Exposed at Work		
		All Subjects	Self-reported	Surrogate[b] reported
Ever[a]	OR	1.4	1.3	2.0
	90% CI	0.6, 3.2	0.5, 3.4	0.7, 5.5
Never	OR	2.2	2.8	2.0
	90% CI	0.5, 9.2	0.4, 20.7	0.3, 13.9

[a]Both current and former smokers included.
[b]Self-reported controls were the comparison group for the surrogate-reported cases.

with multiple logistic models and found to vary with cigarette smoking habits (Table 5). The odds ratios were higher for the never smoking females; and in these never smokers the two exposure variables gave comparable risk estimates.

Discussion

In the context of a population-based case-control study in New Mexico, we have examined the risk of lung cancer associated with marriage to a cigarette smoker. The results indicated increased risk from this exposure in never smokers, but not in active smokers.

Methodologic limitations of the case-control approach for studying the relation between involuntary exposure to tobacco smoke and lung cancer must be considered. Misclassification of both active and passive exposure to cigarette smoke is of particular concern. With regard to active smoking, we assigned exposure on the basis of a comprehensive interview with either the index case or a surrogate respondent. For four of the 28 cases among never smokers, information in the hospital record conflicted with the interview. Because a similar, additional source of data was not available for controls, we did not exclude the four cases from this report. The findings were unchanged, however, when they were removed from the analyses.

We assessed passive exposure to tobacco smoke only from marriage to a smoking spouse; exposures from other smokers at home and in the workplace were not assessed. Thus, subjects may have been misclassified on total passive smoke exposure. Wald and Ritchie[27] have shown that nonsmoking men married to smoking women report greater exposure to the smoke of others outside of the home than nonsmoking men married to nonsmoking women. Wald and Richie suggest that information on smoking by the spouse conveys some information on other sources of exposure.

Surrogate interviews were necessary for 19 of the 28 never smokers. While the validity of surrogate information has been questioned for some exposures,[28] the surrogate respondents were primarily surviving spouses, who provided information on their own smoking habits and those of previous spouses, if any. Extensive misclassification introduced by the surrogate interviews thus appears unlikely, although spouses aware of the putative association of passive smoking with lung cancer may have minimized their own smoking. Spouse surrogates may have supplied more accurate information concerning their own smoking than would have been available from the index subject. The much higher proportion of surrogate interviews for cases than for controls could have introduced differential misclassification and biased effect measures upwards.

The results of the present case-control study complement those from other case-control studies[4,7–10] and from cohort studies,[3,11] which showed increased lung cancer risks in never smokers married to smokers. The magnitude of the effect of marriage to a smoker in the present study, about a two-fold increase in risk (Tables 2 and 3), is comparable to findings by Hirayama[3] and by Akiba, *et al*,[9] in Japan, by Trichopoulos, *et al*,[4] in Greece, and by Correa, *et al*,[7] and by Dalager, *et al*,[10] in the United States. A weak exposure-response relation was present with duration of passive exposure, but not with average number of cigarettes smoked daily by the spouse (Table 4). In contrast, in a larger case-control study, Garfinkle, *et al*,[8] found a trend of increasing risk for nonsmoking women with the number of cigarettes smoked daily at home by their husbands.

In active smokers, we found that residence with a smoker did not elevate lung cancer risk (Table 2). The lack of association in active smokers is consistent with the quantitative differences in the exposures of active and passive smoking.[6] Furthermore, active smokers must receive more passive exposure to tobacco smoke from their own smoking, than from the smoking of others. The odds ratios for passive smoking in active smokers, all at or near unity, provide evidence against consistent under- or overreporting of exposure (Tables 2 and 3).

We also assessed the effects of marriage to a spouse employed in jobs possibly involving contact with asbestos. We hypothesized that asbestos brought into the home by the spouse might increase lung cancer risk in smokers and nonsmokers. Domestic exposure has been previously associated with mesothelioma, pleural abnormalities, and changes in the lung parenchyma.[29] We used both a lifetime occupational history for the spouse of the index case and reported contact with asbestos to assess possible indirect exposure of the cases to asbestos.

With both approaches for determining exposure, we found associated elevations of risk for lung cancer (Table 5). The effect was more evident in never smokers, although comparable relative risks would be anticipated if cigarette smoking and asbestos exposure interact multiplicatively in this setting.[29,30] The magnitude of effect was surprisingly large in view of the range of excess risk found in asbestos-exposed workers and of the results of risk estimation.[29,30]

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1989 Revisions of the US Standard Certificates and Reports

The National Center for Health Statistics (NCHS) has recently distributed to the 50 states the 1989 revisions of the US Standard Certificates and Reports of Live Birth, Death, Fetal Death, Induced Termination of Pregnancy, Marriage, and Divorce. These documents serve as models for the various states to use in developing their own forms. NCHS recommends that revised certificates and reports incorporating the 1989 changes be implemented in all states by January 1, 1989.

The US Standard Certificates and Reports were developed jointly by the NCHS and state vital registration and statistics executives. Advice was obtained from persons and organizations throughout the United States who represented users of vital statistics data and those who complete the documents. The content reflects a consensus of what needs to be collected about each vital event to serve both the legal and statistical uses of these records in the 1900s.

Among the more significant modifications made in these new revisions are:

- the addition of an Hispanic identifier to the live birth and death certificates and the fetal death and induced termination of pregnancy reports;
- changes in the birth certificate and fetal death report to obtain more detailed information about the pregnancy and its outcome; and
- some of the factors that may have improved quality and completeness of the cause of death.

Information about the revision process and copies of the standard certificates and reports can be obtained by writing or calling:

George A. Gay
Chief, Registration Methods Branch
Division of Vital Statistics, NCHS
3700 East-West Highway, Room 1-44
Hyattsville, Maryland 20782
Tel: (301) 436-8815